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Effect of Air Pollution on Heart Failure: Systematic Review and Meta-Analysis

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Background: Heart failure (HF) poses a significant global disease burden. The current evidence on the impact of air pollution on HF remains inconsistent.

Objectives: We aimed to conduct a systematic review of the literature and meta-analysis to provide a more comprehensive and multiperspective assessment of the associations between short- and long-term air pollution exposure and HF from epidemiological evidences.

Methods: Three databases were searched up to 31 August 2022 for studies investigating the association between air pollutants ($PM_{2.5}$, PM_{10}, NO_2, SO_2, CO, O_3) and HF hospitalization, incidence, or mortality. A random effects model was used to derive the risk estimations. Subgroup analysis was conducted by geographical location, age of participants, outcome, study design, covered area, the methods of exposure assessment, and the length of exposure window. Sensitivity analysis and adjustment for publication bias were performed to test the robustness of the results.

Results: Of 100 studies covering 20 countries worldwide, 81 were for short-term and 19 were for long-term exposure. Almost all air pollutants were adversely associated with the risk of HF in both short- and long-term exposure studies. For short-term exposures, we found the risk of HF increased by 1.8% [relative risk (RR) = 1.018, 95% confidence interval (CI): 1.011, 1.025] and 1.6% (RR = 1.016, 95% CI: 1.011, 1.020) per 10-$\mu g/m^3$ increment of $PM_{2.5}$ and PM_{10}, respectively. HF was also significantly associated with NO_2, SO_2, and CO, but not O_3. Positive associations were stronger when exposure was considered over the previous 2 d (lag 0–1) rather than on the day of exposure only (lag 0). For long-term exposures, there were significant associations between several air pollutants and HF with RR (95% CI) of 1.748 (1.112, 2.747) per 10-$\mu g/m^3$ increment in $PM_{2.5}$, 1.212 (1.010, 1.454) per 10-$\mu g/m^3$ increment in PM_{10}, and 1.204 (1.069, 1.356) per 10-ppb increment in NO_2, respectively. The adverse associations of most pollutants with HF were greater in low- and middle-income countries than in high-income countries. Sensitivity analysis demonstrated the robustness of our results.

Discussion: Available evidence highlighted adverse associations between air pollution and HF regardless of short- and long-term exposure. Air pollution is still a prevalent public health issue globally and sustained policies and actions are called for to reduce the burden of HF. https://doi.org/10.1289/EHP11506

Introduction

Heart failure (HF) is a complex syndrome caused by cardiac structural or functional impairment and is often the terminal stage of various cardiovascular diseases (CVDs). Although the age-standardized incidence of HF has been declining since 2000,[1] it remains highly prevalent and contributes to considerable mortality and represents a considerable disease burden globally owing to the aging of the population. In 2017, an estimated 63.4 million people worldwide suffered from HF, representing a 106% increase in the years of lived with disability (YLDs) compared with 1990.[2]

According to the Global Burden of Disease Study in 2019, air pollution in urban and rural areas was responsible for ∼6.6 million premature deaths,[3] mainly as a result of exposure to fine and ultrafine particulate matter with an aerodynamic diameter of ≤2.5 μm ($PM_{2.5}$ and UFP, respectively). Observational and experimental studies have demonstrated that air pollution has a strong impact on CVDs such as coronary artery disease,[4] stroke[5] and myocardial infarction.[6] In a previous meta-analysis that included short-term air pollution exposure studies published through 15 July 2012, air pollution was reported to be associated with HF hospitalization or death within a few days after exposure; however, most of the reports were from high-income countries (HICs) with lower concentrations of air pollutants (e.g., $PM_{2.5}$, concentration ranging from 4.5 to 20.5 $\mu g/m^3$).[7]

Over the past decade, numerous studies have provided new evidence on the link between air pollution and HF from low- and middle-income countries (LMICs) or long-term exposures. Compared with the HICs, the air quality in LMICs is often worse, and many LMICs have annual average $PM_{2.5}$ concentrations well above 20 $\mu g/m^3$.[8,9] A significant positive association between $PM_{2.5}$ concentration and HF hospitalizations was observed in 26 Chinese cities with an average annual $PM_{2.5}$ concentration of 63.5 $\mu g/m^3$.[8] In addition, associations between long-term exposure to air pollution and HF have increasingly been reported.[10–13] However, the results across studies have been largely inconsistent owing to variations in demographic characteristics, study design, types and concentrations of pollutants, and other factors.[10,11] A cohort study in Toronto, Canada, showed that long-term exposure to $PM_{2.5}$ was significantly associated with congestive HF.[12] Conversely, another cohort study of 196,167 adults in South Korea found a significant negative association between long-term $PM_{2.5}$ exposure and HF.[13] Given the availability of new studies, we performed a systematic review and meta-analysis to provide a more comprehensive and multiperspective assessment of the associations of both short- and long-term exposure to particulate and gaseous air pollutants with HF in adults and to provide more valuable information for further studies and prevention strategies.

Methods

Search Strategy and Selection Criteria

This systematic review and meta-analysis was conducted following the 2020 Preferred Reporting Items for Systematic Reviews

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and Meta-Analyses (PRISMA) guideline, and the review methods were prespecified prior to the execution of this review. The objectives of the review were defined according to three elements: *a*) population, including adults ≥18 y old; *b*) exposure, indicating exposure to air pollution or specific air pollutant; and *c*) outcome, hospitalization or mortality related to, or incidence or prevalence of, HF. We searched for studies focusing on the association between air pollution and HF in human from three databases: PubMed, Ovid Medline, and Embase. The primary search keywords were based on "air pollution" and "heart failure." In "air pollution," we included the terms "air pollution" or "air pollutants," "particulate matter ($PM_{2.5}$, PM_{10})," "nitrogen dioxide (NO_2)," "sulphur dioxide (SO_2)," "carbon monoxide (CO)," and "ozone (O_3)." Keywords in "heart failure" were "heart failure," "cardiac failure," "heart decompensation," "myocardial failure," and "cardiac insufficiency." The bibliography lists of eligible studies and relevant reviews were also screened. A literature search was conducted among studies published through 31 August 2022. The complete search strategies are available in the Supplemental Material, "Search Strategies in Different Databases." The retrieval and screening process of the literature was repeated independently by two researchers (J.Y. and L.Z.), and any inconsistencies were discussed and agreed upon with a third researcher (H.Z.).

Epidemiological studies were included if they *a*) presented data and reported the association between exposure to particulate ($PM_{2.5}$, PM_{10}) or gaseous (NO_2, SO_2, CO, O_3) air pollutants and HF; *b*) reported data for emergency department visits, HF hospitalization, HF incidence, HF mortality in all participants regardless of history of HF, or all-cause mortality in patients with HF; *c*) reported odds ratio (OR)/relative ratio (RR)/hazard ratio (HR) and the corresponding 95% confidence interval (CI) for the association between air pollutants and HF; and *d*) were peer-reviewed original studies in English.

Data Extraction

Data extraction of included studies was performed independently by two researchers (J.Y. and L.Z.), and a third researcher (H.Z.) joined the discussion to resolve any disagreements. For each eligible study, we extracted general study characteristics (author, published year, study period, country, data source, study design, sample size, age, population, type of air pollutants, exposure assessment methods) and statistical values (mean or range of exposure to air pollutants, OR/RR/HR and 95% CI, adjusted covariates). For short-term exposure studies, we also extracted the number of lags up to lag 7 if applicable.

Assessing the Risk of Bias

We evaluated the risk of bias for each of the studies followed the Navigation Guide[14,15] across the following domains: recruitment strategy, blinding, confounding, exposure assessment, incomplete outcome data, selective outcome reporting, conflicts of interest, or other resources of bias that could put the study at risk of bias.[15,16] For each domain, risk of bias was rated as "low," "probably low," "probably high," or "high," and the specific detailed rating definitions of each domain were adapted with reference to the Navigation Guide and related literature combining with the topic of our review (general definition of the low risk of bias is shown in Table 1; for detailed criteria, see Supplemental Material, "Instructions for Making Risk of Bias Determinations"). Two researchers (J.Y. and L.C.) independently made and documented risk of bias determinations for each study across all domains. When these two researchers could not reach consensus on a risk of bias domain, another researcher (L.Z.) reviewed the results. If all three researchers were unable to reach agreement on a risk of bias determination for a particular domain, the more conservative judgment was adopted (e.g., if one reviewer made a judgment of low risk and the others made a judgment of probably low risk, the probably low risk judgment was used).

Table 1. Summary of risk of bias domains and criteria for low risk evaluation.

Risk of bias domain[a]	Criteria for low risk evaluation[b]
Recruitment strategy	Protocols for recruitment and inclusion/exclusion criteria applied similarly across study groups.
Blinding	Knowledge of the exposure ensured when assessing outcome, or judgment that outcome measurement not likely to be influenced by lack of blinding.
Exposure assessment	Confidence in the accuracy of the exposure assessment methods that minimizes exposure misclassification, that is, validity and reliability measures specified for monitoring and modeling.
Confounding	All the important potential confounders prespecified are accounted for.
Incomplete outcome	No missing outcome data, balanced attrition across groups, or for continuous outcome data, plausible effect size among missing outcomes not enough to have a relevant impact on the observed effect size.
Selective outcome reporting	All prespecified outcomes outlined in the protocol, methods, abstract, or introduction reported in the prespecified way.
Conflict of interest	The study did not receive support from a company, study author, or other entity having a financial interest in the outcome of the study.
Other bias	The study appears to be free of other sources of bias.

[a]The eight domains of risk of bias assessment were strictly followed the Navigation Guide.[14,15]
[b]The criteria for risk evaluation was adapted from the Navigation Guide combined with the topic of our review.

Data Syntheses

RR was used to represent the association between exposure and outcome uniformly. RRs were pooled after transformation with a consistent increment[7] in pollutant concentrations as follows: 10 $\mu g/m^3$ for $PM_{2.5}$ and PM_{10}; 10 ppb for NO_2 (1 ppb = 46/22.4 $\mu g/m^3$), SO_2 (1 ppb = 64/22.4 $\mu g/m^3$), and O_3 (1 ppb = 48/22.4 $\mu g/m^3$); and 1 ppm for CO (1 ppm = 1,000 × 28/22.4 $\mu g/m^3$). We assumed a linear relationship between exposure and outcome, given that most studies used generalized linear models. Therefore, standardized RRs were calculated for each study using the following formula:

$$RR_{(\text{standardized})} = RR_{(\text{original})}^{\text{Increment(standardized)}/\text{Increment(original)}}.$$

When both associations for hospitalization and mortality of HF in the same population were reported in one study, the effect value of hospitalization with a larger number of events was pooled in the overall risk and they were pooled separately in stratified analysis. One exception is that two long-term studies[17,18] discussed the impact of air pollutants on the all-cause mortality and readmission in patients with HF in the same cohort, and the study with more patients for mortality[17] was included in the meta-analysis.

For several different studies of the same population, we included only the results from time-series analysis (short-term),[19–21] longer follow-up (long-term),[22,23] covering more pollutants,[24,25] or the updated reports[26–30] to estimate the overall risk. Some studies reported stratified risk estimations by location,[11,31] age,[32] and temperature[24,25,33,34] rather than a whole population, and the

stratified results were all incorporated to evaluate the overall risk of HF. For all studies, we pooled the adjusted risk estimates after controlling for confounders such as temporal, meteorological, seasonal, demographic, and socioeconomic factors.

Most short-term exposure studies announced multiple estimates for different lags and were all pooled separately (Excel Table S1), whereas the shortest lag [either single-day (e.g., lag 0) or multiday (e.g., lag 0–1) lag] was pooled for the overall risk. For estimates of different lags, we pooled the effect with at least two estimates separately. Long-term exposure studies were mostly derived from large cohorts and, therefore, the exposure windows were hard to standardize (Excel Table S2). Thus, the most significant result of each study was pooled for overall estimation.

Statistical Analyses

Meta-analyses were conducted only when two or more eligible studies examined the association between the same pollutant and HF. A random effects model was used to estimate the quantitative associations of short-term and long-term exposure to air pollutants with HF risk, which accounted for heterogeneity among studies. Heterogeneity was assessed using I^2 statistics,[35] and an I^2 value $\geq 75\%$ was regarded as considerable heterogeneity. Funnel plots were drawn to assess the asymmetry of studies. The Egger's regression test was used (only when the number of studies was ≥ 10) for testing the publication bias,[36,37] whereas a $p < 0.05$ indicated significant publication bias. We also used the Trim and Fill method to further test and adjust for possible publication bias.[38]

Additional stratified analysis for short-term exposure was carried out by geographical location, age, outcome, study design, covered area, the methods of exposure assessment, and the length of exposure window (each subgroup included at least two studies) to determine whether a potentially susceptible population may exist. For long-term exposure, stratified analyses were done according to covered area, exposure window, exposure stage, and geographical location. Given the limited studies included for long-term exposure to SO_2 and CO, the stratified analysis was not carried out for these exposures. To investigate the source of the heterogeneity, we performed meta-regressions for air pollutants with at least 10 studies included,[16] adjusting several factors such as publication year (continuous), sample size (continuous), population characteristics (general population or CVD patients, age ≥ 65 y old or all age), regions (continent, single or multicity), exposure assessment (monitoring or modeled data), exposure window (only applied in long-term exposure; 1 y or >1 y), outcome definition (only applied in short-term exposure; emergency visit, hospitalization or mortality), and study design (only applied in short-term exposure; time-series, case-crossover or survival analysis). For each pollutant, we selected the meta-regression model with the best R^2 under the premise of convergence as the main model. Sensitivity analysis was used to test the robustness of our results by excluding the studies with maximum or minimum effect size, a special period (i.e., wildfire or storm), small sample size (<10,000 participants), special population (i.e., patients with CVD or HF), and high risk of bias.

The analyses were performed using Stata software (version 16.0; StataCorp). Statistical significance was taken as two-sided $p < 0.05$.

Results

Literature Retrieval and Basic Study Characteristics

The initial searches retrieved 4,403 records from three databases with no additional records found through the bibliography lists, 1,482 records of which were removed for being duplicates and 2,627 records were removed for being irrelevant (Figure S1). Of 294 studies that underwent in-depth full-text review, 100 studies met the inclusion criteria. The eligible studies were divided into two groups according to the interval between the occurrence of exposure and the onset of the outcome. We did not find any eligible studies in which the interval was between 30 d to 1 y. So the studies were grouped as short-term exposure ($n = 81$) if the interval was $\leq$30 d (Table 2), and as long-term exposure ($n = 19$) if the interval was $\geq$1 y (Table 3). For short-term exposure studies, 48 studies used a time-series analysis, 30 studies used a case-crossover analysis, 1 study used both time-series and case-crossover analysis, and 2 cohort studies with survival analysis used the Cox proportion hazard risk model. All 19 long-term studies used the cohort design. No case–control studies investigating these relationships were found in the eligible studies.

All studies covered 20 countries, mostly in the Northern Hemisphere (Figure S2). The top three countries in terms of the number of studies were the United States, China, and Canada, which together accounted for two-thirds of all studies. All included studies covered a wide range of concentrations across several air pollutants; for example, $PM_{2.5}$ ranged from 2.90 to 102.10 $\mu g/m^3$, PM_{10} from 13.00 to 131.50 $\mu g/m^3$, NO_2 from 6.57 to 77.03 ppb, SO_2 from 0.92 to 32.01 ppb, CO from 0.002 to 5.60 ppm, and O_3 from 1.88 to 95.66 ppb. The methods for exposure assessment were mainly based on two types of data sources. One was to estimate individual exposure at the city-/countrywide level based on the real-time monitoring data from national or regional surveillance sites (69 studies of short-term exposure and 3 studies of long-term exposure), whereas the other method was at the individual level based on predicted values of pollutants by various models (12 studies of short-term exposure and 16 studies of long-term exposure), such as the land-use regression model, satellite-based atmospheric aerosol optical depth inversion model, or hybrid model with machine learning.

Short-Term Exposure of Air Pollutants and HF

Significantly positive associations were calculated for exposures to almost all the gaseous and particulate pollutants and HF (Figure 1). A 10-$\mu g/m^3$ increment of $PM_{2.5}$ and PM_{10} was estimated to result in a 1.8% (RR = 1.018, 95% CI: 1.011, 1.025) and 1.6% (RR = 1.016, 95% CI: 1.011, 1.020) increase in risk of HF hospitalization and mortality, respectively. The RRs for gaseous pollutants on HF ranged from 1.010 to 1.037, with all pollutants having an association with increased risk of HF, although the overall risk in O_3 was not significant (RR = 1.010, 95% CI: 0.998, 1.021). In single-day lag models, the strongest associations were generally presented at lag 0, and the strength gradually decreased from lag 0 to lag 7. A clear temporal trend of a gradual reduction in RR was observed within 3 d of exposure, but the adverse impact of pollutants on HF remained for 7 d. Multiday lag models often presented a positive association at lag 0–1, lag 0–2, and lag 0–6 without remarkable temporal tendency. The more apparent positive associations usually appeared at lag 0–1. A 10-ppb increment of O_3 was significantly associated with HF only at lag 0–6 (RR = 1.025, 95% CI: 1.008, 1.042).

Additional stratified analyses were done for short-term exposure studies by outcome, study design, age of participants, geographical location, national economic status, exposure assessment, and covered areas (Figure 2). After grouping by the national economic status (Excel Table S1), the HF risks associated with each pollutant ($PM_{2.5}$, PM_{10}, NO_2, SO_2, CO, and O_3) in LMICs were higher than those in HICs. Compared with Europe and North America, the adverse impacts of pollutants on HF were generally larger in Asia, roughly in line with air pollution levels across continents (Figure 2; Table S1). Studies originating in Oceania

Table 2. Study characteristics of the included studies in short-term exposure (the interval between the occurrence of exposure and the onset of the outcome was ≤30 d) by publication year.

Reference	Country	Publication year	Study year	Data source	Events (N)[a]	Methods	Age (y)	Outcome	Outcome evidence[b]	Pollutants	Lag model	Exposure assessment
Morris et al.[39]	USA	1995	1986–1989	Medicare	227,985	Time series	≥65	HF admission	ICD-9: 428	NO_2, SO_2, CO, O_3	Single-day	Monitoring stations
Schwartz and Morris[40]	USA	1995	1986–1989	Medicare	38,862	Time series	≥65	HF admission	ICD-9: 428	PM_{10}, SO_2, CO, O_3	Multiday	Monitoring stations
Burnett et al.[41]	Canada	1997	1981–1991	Hospital discharge records	157,865	Time series	≥65	HF admission	ICD-9: 427	NO_2, SO_2, CO, O_3	Single-day	Monitoring stations
Poloniecki et al.[42]	UK	1997	1987–1994	Hospital episode records	63,902	Time series	All	HF admission	ICD-9: 428	NO_2, SO_2, CO, O_3	Single-day	Monitoring stations
Morris and Naumova[43]	USA	1998	1986–1989	Medicare	49,674	Time series	≥65	HF admission	ICD-9: 428	PM_{10}, NO_2, SO_2, CO, O_3	Single-day	Monitoring stations
Wong et al.[44]	China	1999	1995–1997	Hospital authority data	NA	Time series	≥65	HF admission	ICD-9: 428	O_3	Single-day	Monitoring stations
Wong et al.[45]	China	1999	1994–1995	Emergency hospital admission data	NA	Time series	All	HF admission	ICD-9: 428	PM_{10}, NO_2, SO_2, O_3	Multiday	Monitoring stations
Burnett et al.[46]	Canada	1999	1980–1994	Hospital discharge records	49,331	Time series	All	HF admission	ICD-9: 428	$PM_{2.5}$, PM_{10}, NO_2, SO_2, CO, O_3	Multiday	Monitoring stations
Lippmann et al.[28]	USA	2000	1992–1994	Medicare	18,615	Time series	≥65	HF admission	ICD-9: 428	$PM_{2.5}$, PM_{10}, NO_2, SO_2, CO, O_3	Multiday	Monitoring stations
Linn et al.[47]	USA	2000	1992–1995	CA OSHPD	53,214	Time series	≥30	HF admission	APR-DRG: 127	PM_{10}, NO_2, CO, O_3	Single-day	Monitoring stations
Goldberg et al.[48]	Canada	2001	1984–1993	Billing and prescription data	16,794	Time series	All	Mortality	ICD-9: 428	$PM_{2.5}$	Multiday	Monitoring stations
Hoek et al.[27]	Netherlands	2001	1986–1994	Death certificates	45,360	Time series	All	Mortality	ICD-9: 428	PM_{10}, NO_2, SO_2, CO, O_3	Single- and multiday	Population-oriented monitoring stations
Kwon et al.[21]	South Korea	2001	1994–1998	Mortality records	1,807	Time series and case-crossover	All	Mortality	ICD-9: 428; ICD-10: I50	PM_{10}, NO_2, SO_2, CO, O_3	Single-day	Monitoring stations
Ye et al.[49]	Japan	2001	1980–1995	Ministry of health and welfare	4,732	Time series	≥65	HF admission	ICD-9: 428	NO_2	Single-day	Monitoring stations
McGowan et al.[50]	New Zealand	2002	1988–1998	Hospital admission data	5,146	Time series	All	HF admission	ICD-9: 428	PM_{10}	Single-day	Monitoring stations
Goldberg et al.[26]	Canada	2003	1984–1993	Billing and prescription data	16,794	Time series	≥65	Mortality	ICD-9: 428	$PM_{2.5}$, NO_2, SO_2, CO, O_3	Single- and multiday	Fixed-site monitoring stations
Koken et al.[51]	USA	2003	1993–1997	Agency for Health Care Research and Quality	1,860	Time series	≥65	HF admission	ICD-9: 428	CO	Single-day	Monitoring stations
Bateson and Schwartz[52]	USA	2004	1988–1991	Medicare	26,923	Time series	≥65	Mortality	ICD-9: 428	PM_{10}	Multiday	Monitoring stations
Metzger et al.[19]	USA	2004	1993–2000	Billing data	20,160	Time series	All	HF admission	ICD-9: 428	$PM_{2.5}$, PM_{10}, NO_2, SO_2, CO, O_3	Multiday	Monitoring stations
Wellenius et al.[53]	USA	2005	1987–1999	Medicare	55,019	Time series	≥65	HF admission	ICD-9: 428.0; 428.1	$PM_{10,}$ NO_2, SO_2, CO, O_3	Multiday	Monitoring stations
Barnett et al.[32]	Australian and New Zealand	2006	1998–2001	Government health departments (Australia) and Ministry of Health (NZ)	27,703	Case-crossover	≥65	HF admission	ICD-9: 428; ICD-10: I50	$PM_{2.5}$, PM_{10}, NO_2, CO	Multiday	Monitoring stations
Dominici et al.[54]	USA	2006	1999–2002	Medicare	986,392	Time series	≥65	HF admission	ICD-9: 428	$PM_{2.5}$	Single-day	Monitoring stations
Martins et al.[55]	Brazil	2006	1996–2001	Department of Data Analysis of the Unified Health System	21,014	Time series	All	HF admission	ICD-10: I50	PM_{10}	Single-day	Monitoring stations
Wellenius et al.[56]	USA	2006	1986–1999	Medicare	55,019	Case-crossover	≥65	HF admission	ICD-9: 428	PM_{10}	Single-day	Monitoring stations
Lee et al.[34]	China	2007	1996–2004	National Health Insurance	13,475	Time series	All	HF admission	ICD-9: 428	PM_{10}, NO_2, SO_2, CO, O_3	Multiday	Monitoring stations
Peel et al.[20]	USA	2007	1993–2000	Billing data	20,073	Case-crossover	≥65	HF admission	ICD-9: 428	PM_{10}, NO_2, SO_2, CO, O_3	Multiday	Monitoring stations
Forastiere et al.[57]	Italy	2008	1997–2004	Regional registers of causes of death	29,707	Case-crossover	≥35	Mortality	ICD-9: 428	PM_{10}	Multiday	Monitoring stations
Pope et al.[58]	USA	2008	1993–2006	Intermountain health care electronic medical records data	2,618	Case-crossover	All	HF admission	ICD-9: 428	$PM_{2.5}$	Multiday	Monitoring stations
Yang[31]	China	2008	1996–2004	Medical insurance file	24,240	Case-crossover	All	HF admission	ICD-9: 428	PM_{10}, NO_2, SO_2, CO, O_3	Multiday	Monitoring stations
Bell et al.[59]	USA	2009	1999–2005	Medicare data	1,142,928	Time series	≥65	HF admission	ICD-9: 428	CO	Single-day	Monitoring stations
Haley et al.[60]	USA	2009	2001–2005	New York Statewide Planning and Research Cooperative System	168,436	Case-crossover	All	HF admission	ICD-9: 428	$PM_{2.5}$	Single-day	Monitoring stations

Table 2. (*Continued.*)

Reference	Country	Publication year	Study year	Data source	Events (N)[a]	Methods	Age (y)	Outcome	Outcome evidence[b]	Pollutants	Lag model	Exposure assessment
Stieb et al.[61]	Canada	2009	1992–2003	Emergency department data	32,313	Time series	All	HF admission	ICD-9: 428; ICD-10: I50	$PM_{2.5}$, PM_{10}, NO_2, SO_2, CO, O_3	Single-day	Monitoring stations
Ueda et al.[62]	Japan	2009	2002–2004	Ministry of Health, Labor and Welfare of Japan	16,928	Time series	All	Mortality	ICD-10: I50	$PM_{2.5}$	Single- and multiday	Monitoring stations
Zanobetti et al.[63]	USA	2009	2000–2003	Medicare data	238,587	Time series	≥65	HF admission	ICD-9: 428	$PM_{2.5}$	Multiday	Monitoring stations
Belleudi et al.[64]	Italy	2010	2001–2005	Regional hospital discharge registry	18,011	Case-crossover	≥35	HF admission	ICD-9: 428	$PM_{2.5}$, PM_{10}	Single- and multiday	Monitoring stations
Colais et al.[65]	Italy	2012	2001–2005	Regional hospital discharge registry	47,011	Case-crossover	≥65	HF admission	ICD-9: 428	PM_{10}	Single- and multiday	Monitoring stations
Hori et al.[66]	Japan	2012	2006–2010	Emergency hospital admission data	204	Time series	All	HF admission	NA	NO_2, SO_2	Single-day	Monitoring stations
Kim et al.[67]	USA	2012	2003–2007	Hospital admission discharge data	14,283	Time series	All	HF admission	ICD-9: 428	$PM_{2.5}$	Single-day	Monitoring stations
Hsieh et al.[11]	China	2013	2006–2010	Medical insurance file	20,776	Case-crossover	All	HF admission	ICD-9: 428	$PM_{2.5}$	Multiday	Monitoring stations
Milojevic et al.[33]	UK	2014	2003–2008	Hospital Episode Statistics and Office for National Statistics	373,538	Case-crossover	All	HF admission and mortality	ICD-10: I50	$PM_{2.5}$, PM_{10}, NO_2, SO_2, CO, O_3	Multiday	Monitoring stations
Talbott et al.[68]	USA	2014	2001–2008	Hospital discharge data	1,493,064	Case-crossover	All	HF admission	ICD-9: 428	$PM_{2.5}$	Single- and multiday	CMAQ-derived predicted daily $PM_{2.5}$ data were used as estimated by the U.S. EPA at the centroid of each Census Bureau ZIP code tabulation area (ZCTA)
Yang et al.[69]	China	2014	2008–2012	Emergency ambulance dispatches	3,375	Time series	All	HF admission	NA	PM_{10}, NO_2, SO_2	Single- and multiday	Monitoring stations
Chang et al.[25]	China	2015	2006–2010	Medical insurance file	7,930	Case-crossover	All	HF admission	ICD-9: 428	$PM_{2.5}$	Multiday	Monitoring stations
Chen et al.[24]	China	2015	2006–2010	Medical insurance file	7,930	Case-crossover	All	HF admission	ICD-9: 428	$PM_{2.5}$, PM_{10}	Multiday	Monitoring stations
Devos et al.[70]	Belgium	2015	2007–2012	Hospital discharge data	1,340	Case-crossover	All	HF admission	ICD-9: 428	$PM_{2.5}$, PM_{10}, NO_2	Multiday	Monitoring stations combined with land cover data obtained from satellite images in a spatial temporal interpolation method
Grineski et al.[71]	USA	2015	2005–2010	Health Care Information Council	7,947	Case-crossover	≥45	HF admission	ICD-9: 428	$PM_{2.5}$, NO_2	Multiday	Monitoring stations
Alessandrini et al.[72]	Italy	2016	2006–2010	Regional Registers of Causes of Death	44,032	Case-crossover	≥65	Mortality	ICD-9: 428	$PM_{2.5}$, PM_{10}	Multiday	Monitoring stations
Dabass et al.[73]	USA	2016	1999–2011	Department of Health Vital Statistics Division	4,358	Case-crossover	All	Mortality	ICD-10: I50	$PM_{2.5}$	Single- and multiday	Spatiotemporal modeling based on air monitors
Vaduganathan et al.[74]	Italy	2016	2004–2007	Hospital discharge database	1,386	Time series	All	HF admission	ICD-9: 428	PM_{10}	Single- and multiday	Monitoring stations
Weber et al.[75]	USA	2016	2004–2006	New York Statewide Planning and Research Cooperative System	114,137	Case-crossover	≥35	HF admission	ICD-9: 428	$PM_{2.5}$	Single- and multiday	Hierarchical Bayesian model
Hsu et al.[10]	USA	2017	1991–2006	Department of Health's Statewide Planning and Research Cooperative System	999,264	Time series	All	HF admission	ICD-9: 428	$PM_{2.5}$	Single-day	Estimating gridded fields of total and speciated fine particulate matter ($PM_{2.5}$) concentrations for time periods and regions not covered by observational data based on hourly meteorologic observations

Table 2. (*Continued.*)

Reference	Country	Publication year	Study year	Data source	Events (N)[a]	Methods	Age (y)	Outcome	Outcome evidence[b]	Pollutants	Lag model	Exposure assessment
Xu et al.[76]	China	2017	2013.1–12	Computerized medical record system	1,241	Time series	All	HF admission	ICD-10: I50	$PM_{2.5}$	Single- and multiday	Monitoring stations
Buteau et al.[77]	Canada	2018	1991–2003	Medicare data	63,534	Case-crossover	≥65	Mortality	ICD-9: 428	NO_2, O_3	Single- and multiday	Spatially-resolved, time-dependent model to predict concentrations based on the fixed-site monitors
Huynh et al.[78]	Australia	2018	2009–2012	Department of Health and Human Services	1,246	Time series	NA	HF admission	ICD-9: 402.x1, 404.x1, 404.x3, 428.x, 428.xx	$PM_{2.5}$	Single- and multiday	Monitoring stations
Krall et al.[79]	USA	2018	2002–2008	Electronic billing data	203,706	Time series	NA	HF admission	ICD-9: 428	$PM_{2.5}$, PM_{10}, NO_2, SO_2, CO, O_3	Single-day	Monitor concentrations were fused with CMAQ model estimates based on monitoring stations
Li et al.[80]	China	2018	2013–2017	Beijing Municipal Commission of Health and Family Planning Information Center	58,393	Case-crossover	All	HF admission	ICD-10: I50	CO	Single- and multiday	Monitoring stations
Li et al.[81]	China	2018	2010–2012	Beijing medical claim data for employees	15,256	Time series	All	HF admission	ICD-10: I50	$PM_{2.5}$	Single- and multiday	Monitoring stations
Liu et al.[8]	China	2018	2014–2015	National Health Care Data Center	105,501	Case-crossover	All	HF admission	ICD-10: I50	$PM_{2.5}$, PM_{10}, NO_2, SO_2, CO	Single- and multiday	Monitoring stations
Pearce et al.[82]	USA	2018	2002–2013	The South Carolina Revenue and Fiscal Affairs Office of Health Statistics	184,034	Time series	All	HF admission	ICD-9: 428	$PM_{2.5}$, NO_2, SO_2, O_3	Single- and multiday	Monitoring stations
Ran et al.[83]	China	2018	2011–2014	Hospital Authority Corporate Data Warehouse of Hong Kong	54,003	Time series	All	HF admission	ICD-9: 428	$PM_{2.5}$, NO_2, CO, O_3	Multiday	Monitoring stations
Zhang et al.[84]	USA	2018	2005–2016	Hospital admission data	448,222	Case-crossover	≥18	HF admission	ICD-9: 428; ICD-10: I42, I50, I51	$PM_{2.5}$	Single- and multiday	Monitoring stations
Amsalu et al.[85]	China	2019	2013–2017	Beijing Public Health Information Center	58,393	Time series	All	HF admission	ICD-10: I50	$PM_{2.5}$, NO_2, SO_2, CO, O_3	Single- and multiday	Monitoring stations
Feng et al.[86]	China	2019	2013.1–12	Beijing medical research data	1,241	Time series	All	HF admission	ICD-10: I50	PM_{10}	Single- and multiday	The Beijing Environmental Protection Bureau
Johnston et al.[87]	Australia	2019	2009–2014	Emergency ambulance dispatches	21,381	Case-crossover	All	HF admission	NA	$PM_{2.5}$	Single-day	Random forest model inversion of atmospheric aerosol depth data based on satellite remote sensing
Leiser et al.[88]	USA	2019	1999–2009	Center for Medicare and Medicaid Services	8,378 patients with HF, 1,420 deaths	Survival analysis	≥65	Mortality	ICD-9: 428	$PM_{2.5}$	Single- and multiday	Inverse distance weighting estimation of the data from all reporting monitoring stations
Pennington et al.[89]	USA	2019	1998–2010	Emergency department data	79,693	Time series	All	HF admission	ICD-9: 428	$PM_{2.5}$	Single-day	Monitoring stations
Pothirat et al.[90]	Thailand	2019	2016–2017	Emergency and hospitalization visit data	325	Time series	All	HF admission	ICD-10: I50	$PM_{2.5}$, PM_{10}, NO_2, SO_2, CO, O_3	Single-day	Monitoring stations
Tian et al.[91]	China	2019	2014–2017	National database of urban employee basic medical insurance	187,969	Time series	≥18	HF admission	ICD-10: I50	$PM_{2.5}$	Single- and multiday	Average concentration of all monitoring stations in each city (for each which has one to 17 monitoring stations)

Table 2. (*Continued.*)

Reference	Country	Publication year	Study year	Data source	Events (N)[a]	Methods	Age (y)	Outcome	Outcome evidence[b]	Pollutants	Lag model	Exposure assessment
Wu et al.[92]	China	2019	2014–2019	China Center for Disease Control and Prevention	1,782	Time series	All	Mortality	ICD-10: I50	$PM_{2.5}$, PM_{10}	Single- and multiday	Average concentration of all monitoring stations in Lanzhou
de Aguiar Pontes Pamplona et al.[9]	Brazil	2020	2000–2013	Hospital admission data	87,015	Time series	≥60	HF admission	ICD-10: I50	PM_{10}, NO_2, SO_2, CO, O_3	Single-day	Monitoring stations
Nhung et al.[93]	Vietnam	2020	2011–2016	Electronic hospitalization data	11,708	Case-crossover	All	HF admission	ICD-10: I50	$PM_{2.5}$, PM_{10}, SO_2, CO, O_3	Multiday	Fixed monitoring stations in three provinces
Qiu et al.[94]	UK	2020	2000–2012	MEDPAR records	204,774	Case-crossover	≥65	HF admission	ICD-9: 428	$PM_{2.5}$, O_3	Single- and multiday	Hybrid model
Stafoggia et al.[95]	Italy	2022	2013–2015	Hospital discharge records	471,042	Time series	All	HF admission	ICD-9: 428	$PM_{2.5}$, PM_{10}	Multiday	Hybrid spatiotemporal random forest model
Li et al.[96]	China	2021	2015–2020	Hospital admission data	10,466	Time series	All	HF admission	ICD-10: I50.9	$PM_{2.5}$	Single- and multiday	Monitoring stations
Lee et al.[97]	South Korea	2021	2008–2016	National Health Insurance	142,490	Time series	≥65	HF admission	ICD-10: I50.0, I50.1, I50.9	$PM_{2.5}$, PM_{10}, NO_2, SO_2, CO, O_3	Single- and multiday	Six air quality monitoring stations
Leili et al.[98]	Iran	2021	2015–2019	Hospital information system	1,204	Time series	≥18	HF admission	ICD-10: I50.9	$PM_{2.5}$	Single-day	Monitoring stations of the Hamadan's Department of the Environment
Saucy et al.[99]	Switzerland	2021	2000–2015	National mortality records	1,753	Case-crossover	>30	Mortality	ICD-10: I50	$PM_{2.5}$, NO_2	Single- and multiday	Four-stage modeling strategy
Zhao et al.[100]	China	2021	2017–2018	Shenyang Emergency Center	2,560	Time series	NA	HF admission	ICD-10: I50	$PM_{2.5}$	Single- and multiday	Monitoring stations
Mahsin et al.[101]	Canada	2021	2015	The Practitioner Billing Claims database	7,796	Time series	All	HF admission	ICD-9: 428	$PM_{2.5}$	Multiday	Monitoring stations
Wyatt et al.[102]	USA	2022	2004–2016	Electronic health records in the University of North Carolina Healthcare System	17,674	Survival analysis	All	HF admission	ICD-9: 428.x; ICD-10: I50.x	$PM_{2.5}$	Single-day	Hybrid exposure prediction model
Yen and Chen[103]	China	2022	2017–2018	Taiwan's National Health Insurance	979,979	Case-crossover	All	HF admission	ICD-10: I50; received echocardiography examination and had elevated serum NT-proBNP	$PM_{2.5}$, PM_{10}	Single- and multiday	Monitoring stations

Note: APR-DRG, All Patients Refined Diagnosis Related Groups; CA OSHPD, California Office of Statewide Health Planning and Development; CMAQ, Community Multi-Scale Air Quality; CO, carbon monoxide; EPA, Environmental Protection Agency; HF, heart failure; ICD, *International Classification of Disease*; MEDPAR, medical fee for service for parts A & B; NA, not applicable; NO_2, nitrogen dioxide; NT-proBNP, N-terminal pro-brain natriuretic peptide; O_3, ozone; $PM_{2.5}$, particulate matter with the diameter ≤2.5 μm; PM_{10}, particulate matter with the diameter ≤10 μm; SO_2, sulfur dioxide; UK, the United Kindom; USA, the United States.

[a]Number of events, when not stated in the paper, were estimated from mean daily values and the study period.

[b]ICD-9[104] and ICD-10[105] refers to the 9th and 10th revision of *International Classification of Disease*. ICD-9: 428 and ICD-10: I50 both indicated HF, and the suffix of the code indicates the subtype of HF, such as congestive HF, left heart failure, etc.

Table 3. Study characteristics of the included studies in long-term exposure (the interval between the occurrence of exposure and the onset of the outcome was ≥1 y) by publication year.

Reference	Country	Publication year	Study year	Data source	Participants (*N*) and events (*N*)	Age (y)	Outcome	Outcome evidence[a]	Pollutants	Exposure assessment
Kim et al.[106]	Korea	2017	2007–2013	National Health Insurance Service-National Sample Cohort	136,094 participants, 652 patients with HF	≥18	HF incidence	ICD-10: I11.0, I13.0, I13.2, I25.5, I42, I50, O90.3	$PM_{2.5}$, CO, SO_2, NO_2, O_3	Monitoring stations in Seoul collected from the Korean Ministry of Environment website
Sorensen et al.[107]	Denmark	2017	1993–1997	Nationwide Danish National Patient Register	50,935 participants, 2,550 HF new-onset	50–64	HF incidence	ICD-8: 4270–4271; ICD-10: I50, I11.0, I42.0, I42.9	NO_2	Danish AirGIS dispersion modeling system for calculating exposure the annual levels for all participants' addresses
Stockfelt et al.[108]	Sweden	2017	1990–2011	Swedish National Register on Cause of Death and Swedish National Hospital Discharge Register	PPS: 5850 participants (828 patients with HF); GOT-MONICA: 4500 participants (97 patients with HF)	PPS: 64–75 GOT-MONICA: 25–64	HF incidence	ICD-9; ICD-10	$PM_{2.5}$, PM_{10}	High-resolution dispersion model
Downward et al.[109]	Netherlands	2018	1993–2010	National Death Registry of Statistics Netherlands and Hospital Discharge Diagnosis database	33,831 participants, 369 patients with HF	20–70	HF incidence	ICD-9; ICD-10	$PM_{2.5}$, PM_{10}, NO_2	Land-use regression to model pollutants based on the monitoring stations
Bai et al.[22]	Canada	2019	2001–2015	Hospital discharge database	5,062,146 participants, 422,625 patients with HF	35–85	HF incidence	ICD-9; ICD-10	$PM_{2.5}$, NO_2, O_3	$PM_{2.5}$: a global atmospheric chemistry transport model, combined with a geographically weighted regression model to estimate exposure using the satellite retrieved of aerosol optical depth data; NO_2: a national land-use regression model O_3: air quality model outputs from the Canadian and Hemispheric Regional Ozone and NO_x System
Bai et al.[23]	Canada	2019	1996–2012	Hospital discharge database	1,112,060 participants, 106,644 patients with HF	≥30	HF incidence	ICD-9; ICD-10	NO_2	$PM_{2.5}$: a global atmospheric chemistry transport model, combined with a geographically weighted regression model to estimate exposure using the satellite retrieved of aerosol optical depth data; NO_2: land-use regression model
Danesh Yazdi et al.[110]	USA	2019	2000–2012	MEDPAR records	11,084,660 participants, 1,881,452 HF new-onset	All	HF incidence	ICD-9: 428	$PM_{2.5}$, O_3	Hybrid model based on satellite remote sensing, land-use and chemical transport models
Afoakwah et al.[111]	Australia	2020	2010–2015	Hospital visit data	75,140 participants, 6,753 deaths due to HF	≥18	Mortality	ICD-10: I50	PM_{10}, CO	Monitoring stations across the state
Kazemiparkouhi et al.[112]	USA	2020	2000–2008	Center for Medicare and Medicaid Services	22,159,190 participants, 158,649 deaths due to HF	≥65	Mortality	ICD-10: I50	O_3	O_3: monitoring stations; $PM_{2.5}$: GIS-based spatiotemporal model; NO_2: land-use model
Kim et al.[13]	South Korea	2020	2007–2015	National Health Insurance Service-National Sample Cohort	196,167 participants, 3,033 HF onset	30–84	HF incidence	ICD-10: I50	$PM_{2.5}$, PM_{10}	PM_{10}: a pointwise prediction model; $PM_{2.5}$: predict district-specific annual average $PM_{2.5}$ concentrations by using ratios of $PM_{2.5}$ to PM_{10} and PM_{10} predictions
Ward-Caviness et al.[17]	USA	2020	2004–2016	Carolina Data Warehouse for Health	23,302 patients with HF, 4,496 all-cause deaths	≥20	Mortality	ICD-9: 428; ICD-10: I50	$PM_{2.5}$	Multiple machine learning algorithms using monitoring data, satellite measurements, land-use regression variables and other predictors

Table 3. (*Continued.*)

Reference	Country	Publication year	Study year	Data source	Participants (*N*) and events (*N*)	Age (y)	Outcome	Outcome evidence[a]	Pollutants	Exposure assessment
Wang et al.[113]	UK	2021	2006–2010	Health episode statistics and morbidity records	432,530 participants, 4,201 patients with HF	40–69	HF incidence	ICD-10: I11.0, I13.0, I13.2, I50.0, I50.1, I50.9	$PM_{2.5}$, PM_{10}, NO_2	Land-use regression model
Shi et al.[114]	China	2021	2016–2018	Self-reported with medical records or death certificate	4,866 patients with HF, 1,577 readmissions	≥18	HF readmission	NA	$PM_{2.5}$, PM_{10}, NO_2, SO_2, CO, O_3	Monitoring stations of the National Air Pollution Monitoring System
Ward-Caviness et al.[18]	USA	2021	2004–2016	Electronic health record	20,920 patients with HF, 442,244 readmissions, 1,739 7-d readmissions, 7,114 30-d readmissions	All	HF readmission	ICD-9; ICD-10	$PM_{2.5}$	Ensemble machine learning model that incorporates multiple machine learning algorithms to integrate land-use variables, meteorology, chemical transport models, ground-based monitoring, and aerosol optical depth measurements from satellites to estimate $PM_{2.5}$ concentrations at a 1-km^2 resolution for the continental United States
Zhang et al.[12]	Canada	2021	2001–2016	Population-based health administrative database	803,894 participants, 69,801 deaths due to HF	40–85	Mortality	NA	$PM_{2.5}$, NO_2	NO_2: land-use regression model; $PM_{2.5}$: a combination of satellite and chemical transport model
Eum et al.[115]	USA	2021	2001–2008	The Centers for Medicare and Medicaid Services Medicare Enrollment file	49,712,702 participants, 392,034 deaths due to HF	≥65	Mortality	NA	NO_2	NO_2: land-use regression model; $PM_{2.5}$: spatiotemporal smoothing models
Lim et al.[116]	Denmark	2021	1990–2014	Danish Nurse Cohort	22,189 participants, 484 patients with HF	>44	HF incidence	ICD-8: 4270–4271; ICD-10: I50, I11.0, I42.0, I42.9	$PM_{2.5}$, NO_2	The Danish Air Pollution Modeling System
Carlsen et al.[117]	Sweden	2022	1991–1994	The National Board of Health and Welfare's Swedish National Patient Register and Swedish Cause of Death Register and the Uppsala Clinical Research Center's Swedish Coronary Angiography and Angioplasty Register	30,092 participants, 1,323 patients with HF	All	HF incidence	ICD-9: 428; ICD-10: I50, I11.0	$PM_{2.5}$, PM_{10}	Gaussian dispersion model (AERMOD)
Zou et al.[118]	UK	2022	2006–2021	The UK Biobank	321,672 participants, 8,212 cases of incident HF	37–73	HF incidence	Self-reported information, and linkages to a range of health-related records, including primary care, hospital inpatient, and death registry records	$PM_{2.5}$	Land-use regression model

Note: CO, carbon monoxide; GIS, Geographical Information System; GOT-MONICA, the Gothenburg part of the Multinational Monitoring of Trends and Determinants in Cardiovascular Diseases project; HA, hospital admission; HF, heart failure; ICD, *International Classification of Disease*; MEDPAR, medical fee for service for parts A & B; NA, not applicable; NO_2, nitrogen dioxide; O_3, ozone; $PM_{2.5}$, particulate matter with the diameter ≤2.5 μm; PM_{10}, particulate matter with the diameter ≤10 μm; PPS, the Primary Prevention Study cohort; SO_2, sulfur dioxide; UK, the United Kindom; USA, the United States.

[a]ICD-8,[119] ICD-9,[104] and ICD-10[105] refers to the 9th and 10th revision of *International Classification of Disease*. ICD-9: 428 and ICD-10: I50 both indicated HF, and the suffix of the code indicates the subtype of HF, such as congestive HF, left heart failure, etc.

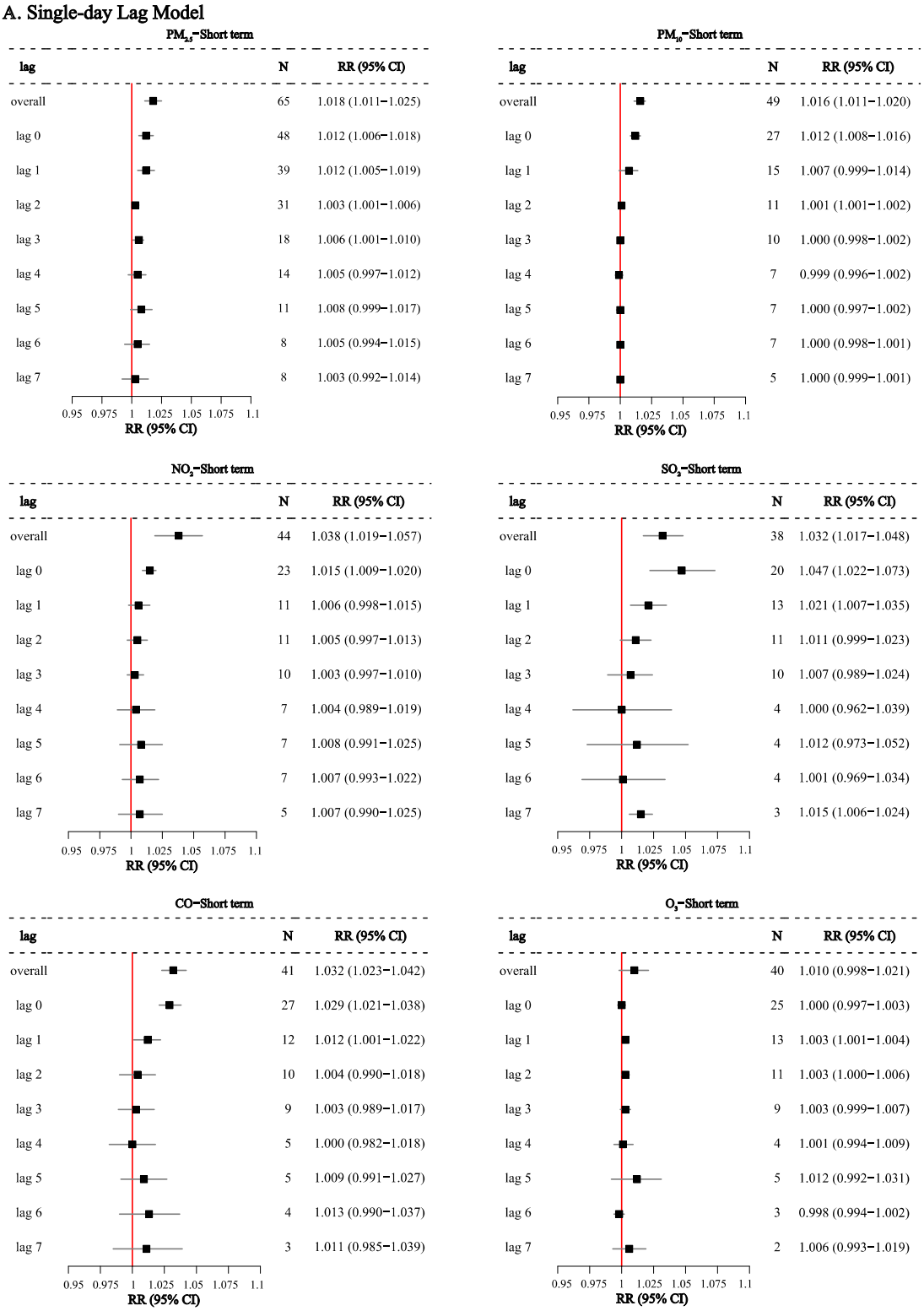

Figure 1. Associations between short-term exposure to air pollutants and heart failure with different lags. Short-term exposure refers to the interval between the occurrence of exposure and the onset of the outcome being ≤30 d. The pooled calculated RRs are plotted as squares, with the corresponding CIs plotted as lines through the squares. (A) presents the pooled results in single-day lag model from lag 0 to lag 7. (B) presents the pooled results in different multiday lag models from lag 0–1 to lag 0–7. The "Overall" RR represents the pooled results of the shortest lag in each study. The increments of each pollutant are 10 μg/m^3 for $PM_{2.5}$ and PM_{10}; 10 ppb for SO_2, NO_2, and O_3; and 1 ppm for CO. The original data for the figure plotting are listed in Table S5. Note: CI, confidence interval; CO, carbon monoxide; N, number of estimations; NO_2, nitrogen dioxide; O_3, ozone; $PM_{2.5}$, particulate matter with the diameter ≤2.5 μm; PM_{10}, particulate matter with the diameter ≤10 μm; RR, relative risk; SO_2, sulfur dioxide.

B. **Multiday Lag Model**

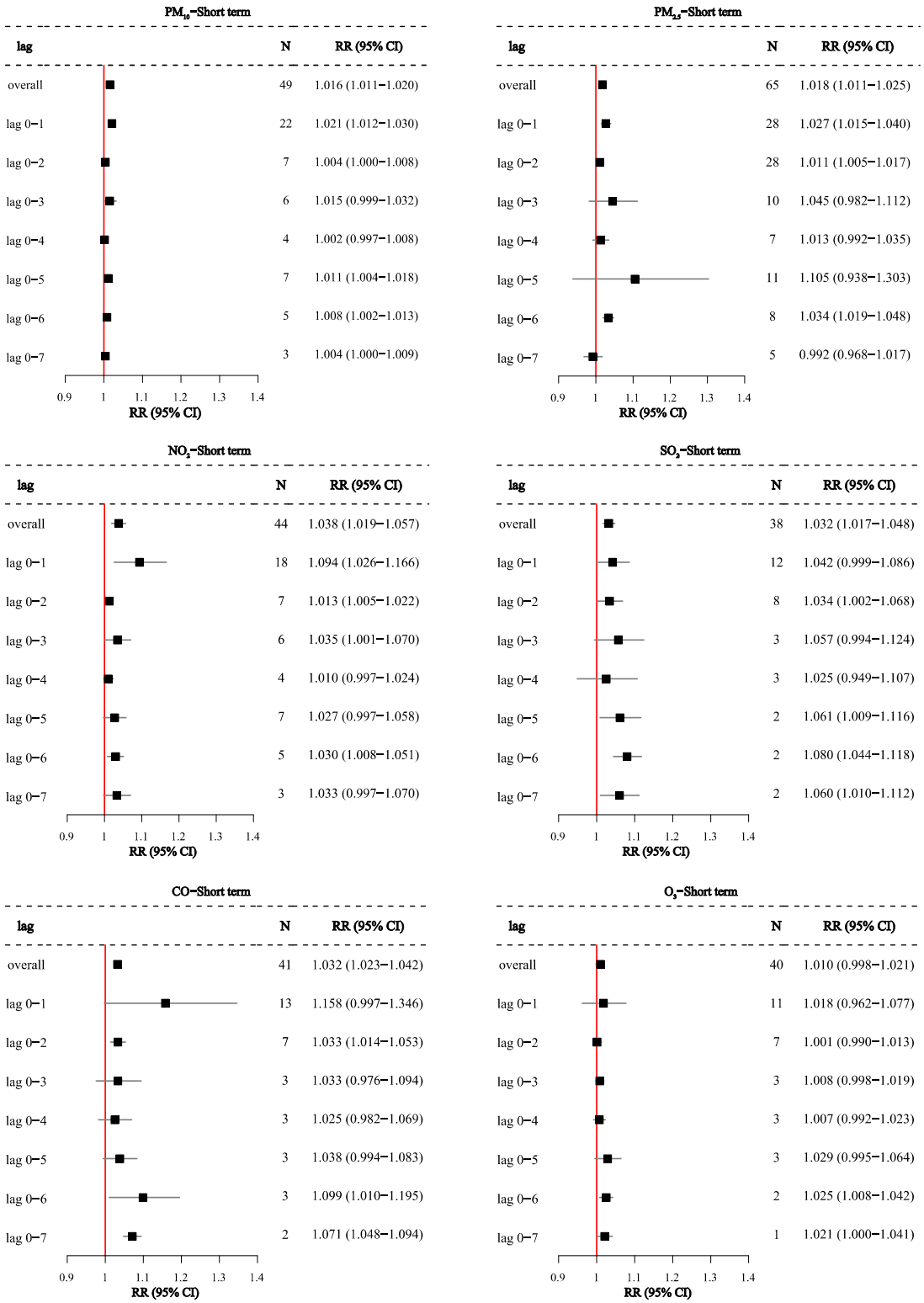

Figure 1. (*Continued.*)

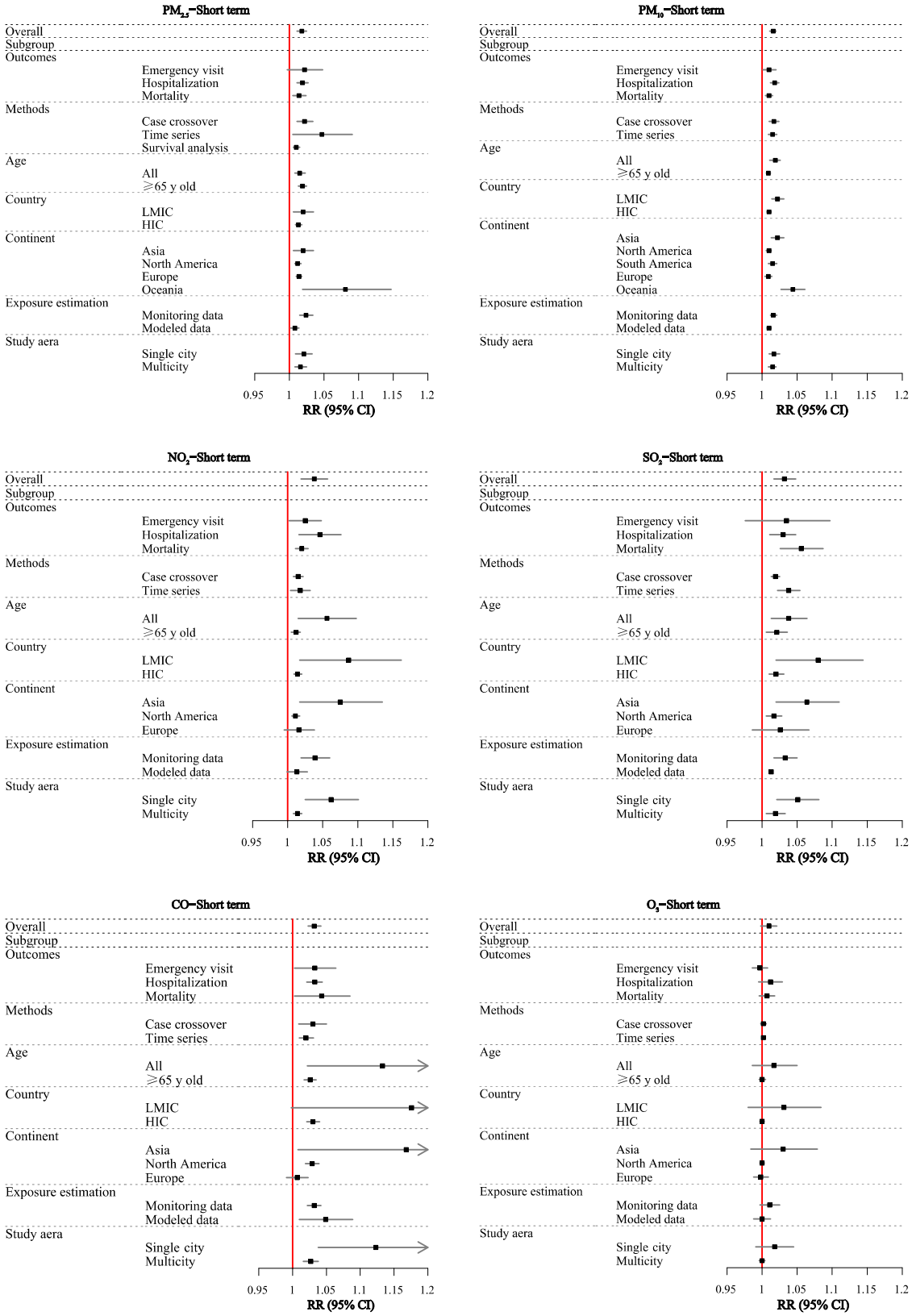

Figure 2. Stratified analyses for short-term exposure studies. Short-term exposure refers to the interval between the occurrence of exposure and the onset of the outcome being ≤30 d. Seven major stratified analyses were performed for each air pollutant. Results were grouped by outcomes, methods, age of population, national economic levels, continents, exposure assessment, and covered area (for details, see Table 2 and Excel Table S1). The pooled calculated RRs of each group are plotted as squares, and the corresponding CIs plotted as lines through the squares. Confidence intervals beyond the *x*-axis range are indicated by arrows. The increments of each pollutant are 10 $\mu g/m^3$ for $PM_{2.5}$ and PM_{10}; 10 ppb for SO_2, NO_2 and O_3; and 1 ppm for CO. The subgroup was included in the stratified analysis only if there were at least two estimations in the subgroup. The original data for the figure plotting are listed in Table S6. Note: CI, confidence interval; CO, carbon monoxide; HIC, high-income country; LMIC, low- and middle-income country; NO_2, nitrogen dioxide; O_3, ozone; $PM_{2.5}$, particulate matter with the diameter ≤2.5 μm; PM_{10}, particulate matter with the diameter ≤10 μm; RR, relative risk; SO_2, sulfur dioxide.

evaluated only the associations of $PM_{2.5}$, and the combined risk of HF hospitalizations and mortality (RR = 1.081, 95% CI: 1.019, 1.147) was higher than that reported on other continents. For most pollutants, using data from monitoring stations to assess the exposure level generally yielded larger effect sizes than using the modeled data (Figure 2; Excel Table S1). There were similar associations between studies conducted in single and multiple cities. The two main methods used in these studies, time-series analysis and case-crossover analysis, also showed similar results.

Long-Term Exposure of Air Pollutants and HF

Among 19 cohort studies assessing the associations of long-term exposure to air pollutants with HF, 14 studies assessed the associations for $PM_{2.5}$, 8 for PM_{10}, 10 for NO_2, 5 for O_3, 3 for CO, and 2 for SO_2 (Table 3). Because of the various exposure windows chosen in the long-term studies, the most significant estimations of each study were synthesized in meta-analysis using the random effects model. Pooled positive associations were calculated for particulate matter ($PM_{2.5}$ and PM_{10}) and NO_2 with HF incidence and mortality (Figure 3). A 10-$\mu g/m^3$ increment in $PM_{2.5}$ and PM_{10} was associated with a 74.8% (RR = 1.748, 95% CI: 1.112, 2.747) and 21.2% (RR = 1.212, 95% CI: 1.010, 1.454) increased overall risk of HF, respectively. A 10-ppb increment in NO_2 was also associated with a 20.4% (RR = 1.204, 95% CI: 1.069, 1.356) higher risk of HF. Neither CO nor O_3 was significantly related to HF. In most eligible original studies included, a positive association between CO and HF was found but not statistically significant. The association between O_3 and HF was controversial across the original studies, with a maximum RR of 1.255 (95% CI: 1.243, 1.268) and a minimum RR of 0.755 (95% CI: 0.709, 0.805). There was a significant adverse association between SO_2 and HF reported in a study from South Korea[13] (RR = 15.316, 95% CI: 8.653, 27.474, per 10-ppb increment), but not in one from China[114] (RR = 1.041, 95% CI: 0.962, 1.125, per 10-ppb increment). Additional stratified analyses were performed by time of exposure window (1 y, ≥1 y), exposure stage (exposure window was prior to the outcome occurrence, exposure window was prior to the baseline), covered area (single city, multicity), and geographic location (Asia, North America, Europe) with at least two studies in each group, and similar estimations were found among all groups in $PM_{2.5}$, NO_2, and O_3 exposure (Figure 4).

Risk of Bias Assessment

Based on our assessment, all 81 studies for short-term exposure and 19 studies for long-term exposure were generally at low risk of bias (Figure 5A,B). According to the Navigation Guide criteria, exposure assessment, confounding, and conflict of interest were identified as the most common sources of risk of bias (Figure 5C). Different pollutant exposures were mostly assessed by the same method in the same study, we assessed the overall risk of bias for each study rather than each pollutant reported within a given study (Figure 5; Excel Tables S1 and S2). When exposure assessment methods were inconsistent among different pollutants in the same study, the risks of bias of exposure assessment were assessed separately, and no inconsistency of the risk of bias rating was found for different pollutants.

Sensitivity Analyses

Sensitivity analyses showed that results were generally robust for both short- and long-term exposure when we excluded studies with the largest or smallest estimated effect size, small sample size, and high risk of bias in any domain (Table S2). Sensitivity analysis after excluding short-term exposure studies that focused only on a special population (i.e., patients with CVD or HF)[26,78,88,102] or a special period,[99,101] such as a wildfire or storm, still supported the soundness of the results (Table S2). For instance, after excluding studies that focused only on special populations, short-term exposure to $PM_{2.5}$ was still associated with a significantly increased risk of HF (RR = 1.017, 95% CI: 1.010, 1.023).

Meta-Regression, Heterogeneity, and Publication Bias Analyses

We observed significant heterogeneity of included studies across all pollutants in both short- and long-term exposure. The meta-regression for short-term exposure to six pollutants showed that none of the investigated factors was a substantial source of the heterogeneity (Table S3). Differences in sample size ($p < 0.05$), exposure assessment ($p < 0.05$), exposure window ($p < 0.01$), population selected ($p < 0.01$), and continent ($p < 0.01$) could explain some of the heterogeneity of long-term $PM_{2.5}$ exposure, but only year of publication ($p < 0.001$) and sample size ($p < 0.01$) appeared to partly explain the heterogeneity in long-term association of NO_2 and HF (Table S4).

For short-term association of $PM_{2.5}$, no evident publication bias was found at the test level of 0.1. For the other five pollutants, although there were apparent publication biases, the results of short-term associations experienced only subtle fluctuations after adjusting for publication bias (Table 4; Figure S3). No significant publication bias was found in the long-term exposure study at the same test level, and the results had no material change after adjustment for publication bias (Table 4; Figure S3). Overall, the effect sizes adjusted for publication bias were generally larger than the original estimates, except long-term exposure to O_3.

Discussion

This study synthesized more evidence of exposure to air pollutants with HF. Both short- and long-term exposure to $PM_{2.5}$, PM_{10}, and NO_2 were estimated to be significantly associated with higher risk of HF. Only short-term exposure to SO_2 and CO had remarkable associations with HF, but not long-term exposure. Neither short- nor long-term exposure to O_3 was significantly associated with an increased risk of HF.

As an extension of the previous study by Shah et al.,[7] we summarized more evidence on short-term exposure from more LMICs and with different exposure measurements and multiple lags. As expected, short-term exposure to almost all pollutants except O_3 were associated with risk of HF with a longer lag time. The HF risks of all gaseous pollutants were higher in this study than those reported in the previous review;[7] for example, we obtained a 1.038 overall risk of short-term exposure to NO_2, which is larger than the risk previously reported (RR = 1.017).[7] Positive associations were stronger when exposure was considered over the previous 2 d (lag 0–1) rather than on the day of exposure only (lag 0), which was inconsistent with the results of the strongest associations at lag 0 in the previous meta-analysis, especially when exposed to particulate matter. In terms of pollutant exposure, the short-term studies included in our meta-analysis covered a wider range of concentrations and had larger medians of average concentrations in $PM_{2.5}$, PM_{10}, SO_2, and O_3.

Although most studies were conducted in the United States, evidence from China, Brazil, and other LMICs in our study has greatly emerged in the recent decade ($\sim$33% of all evidence published after 2012). A higher risk of HF associated with exposure to air pollutants was found in the LMICs than in the HICs, which may be partially due to the higher pollutant levels in LMICs. Among all the included studies, the $PM_{2.5}$ concentrations in nearly half of those from HICs have already met the 2021 standard of the World

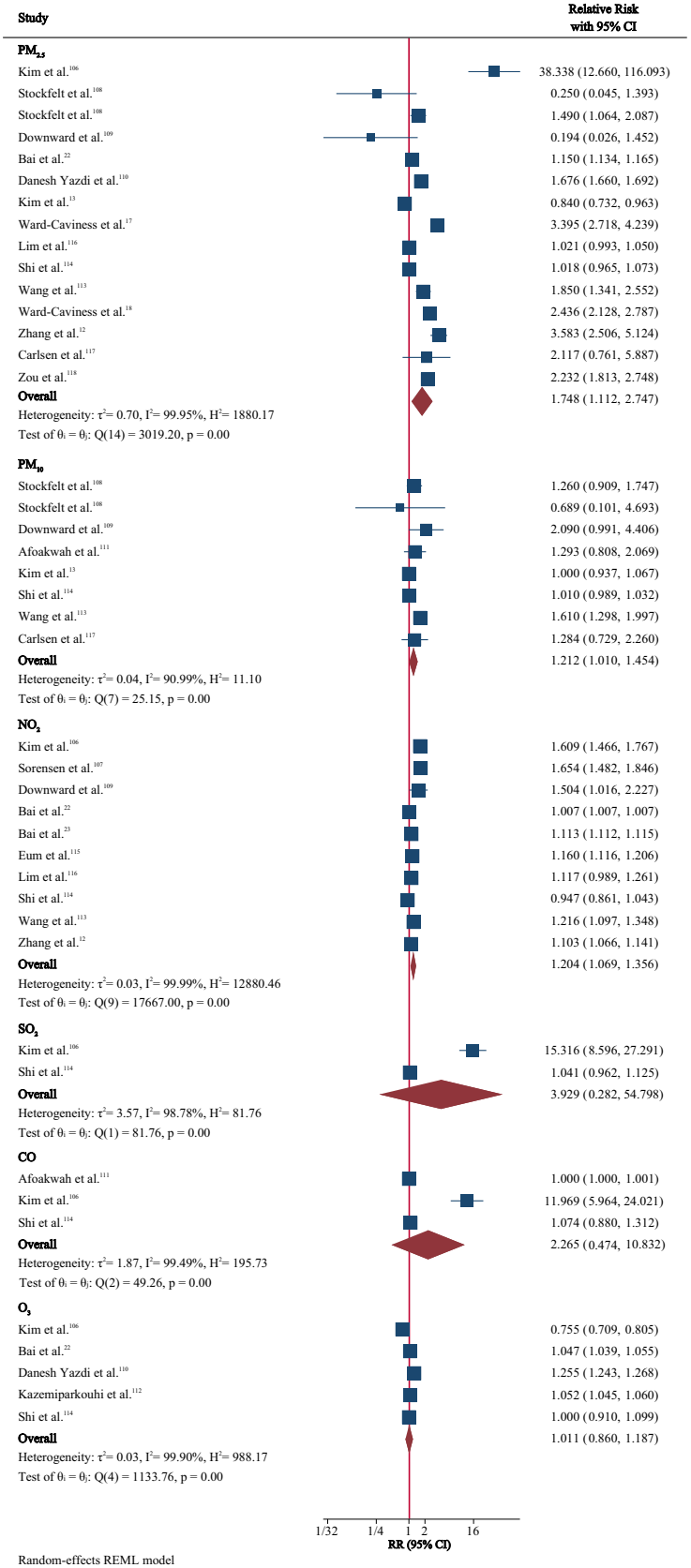

Figure 3. Associations between long-term exposure to air pollutants and heart failure. Long-term exposure refers to the interval between the occurrence of exposure and the onset of the outcome being ≥1 y. Owing to the various exposure window chosen in long-term studies, the most significant estimations of each study were synthesized in meta-analysis using the random effects model. Each square represents the reported effect size in each original study, and the size of the square represents the pooled weight that study was given according to the sample size. The line through the square indicates the corresponding CI. Diamonds represent the overall effect of each pollutant after inclusion in the pooled studies. Note: CI, confidence interval; CO, carbon monoxide; NO_2, nitrogen dioxide; O_3, ozone; $PM_{2.5}$, particulate matter with the diameter ≤2.5 μm; PM_{10}, particulate matter with the diameter ≤10 μm; RR, relative risk; SO_2, sulfur dioxide.

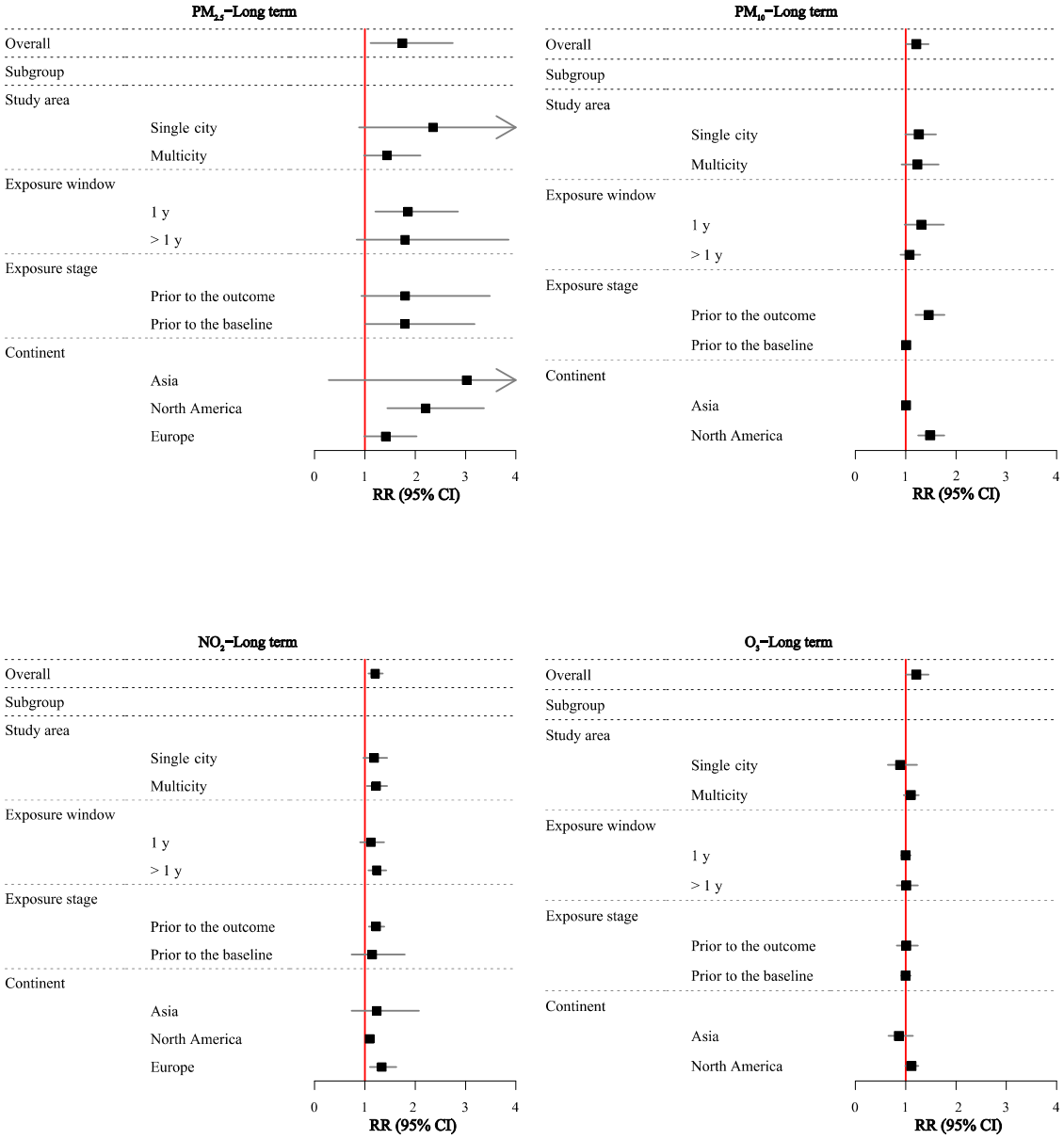

Figure 4. Stratified analysis for long-term exposure studies. Long-term exposure refers to the interval between the occurrence of exposure and the onset of the outcome being ≥1 y. Four major stratified analyses were performed for each air pollutant. Results were grouped by outcomes, methods, age of population, national economic levels, continents, and covered area (for details, see Table 3 and Excel Table S2). The pooled calculated RRs of each group are plotted as squares, and the corresponding CIs plotted as lines through the squares. The increments of each pollutant are 10 $\mu g/m^3$ for $PM_{2.5}$ and PM_{10}, and 10 ppb for NO_2 and O_3. The subgroup was included in the stratified analysis only if there were at least two estimations in the subgroup. The original data for the figure plotting are listed in Table S7. Note: CI, confidence interval; NO_2, nitrogen dioxide; O_3, ozone; $PM_{2.5}$, particulate matter with the diameter ≤2.5 μm; PM_{10}, particulate matter with the diameter ≤10 μm; RR, relative risk.

Health Organization Global Air Quality Guidelines (an average of 24-h <15 $\mu g/m^3$),[120] whereas no studies from LMICs reached the standard. Besides, such differences might also be due to some other inherent and historical discrepancies between LMICs and HICs, including the types of primary pollutant, unique component(s) of pollutants, the epidemiologic status of HF, or the popularity of other confounding factors. Apart from using the traditional monitoring station data, some studies used exposure measurements based on various models that generally had higher spatial and temporal accuracy and could more precisely assess individual exposure levels. However, such methods were usually applied in HICs, which might be related to the level of economy and technology. In LMICs, only one long-term study from China used model-predicted exposure estimates.[114]

The potential mechanisms have been proposed in recent studies for the associations of air pollutants with HF. It has been widely shown using *in vitro*,[121] *in vivo*,[122] and epidemiological[123] studies that ambient air pollutant exposure is associated with oxidative stress, systemic inflammation, and autonomic imbalance. It is proposed that inhaled air pollutants promote oxidative stress and then activate the inflammatory response. Reactive oxygen species released by macrophages and endothelial cells during inflammation further promote the inflammatory response.[124] The hypothalamic–pituitary–adrenal axis could also be activated when exposed to air pollutants, triggering an increase in stress hormone secretion.[125] These physiological changes can lead to increased blood pressure and decreased cardiac output, which, over time, may further result in the progression of endothelial

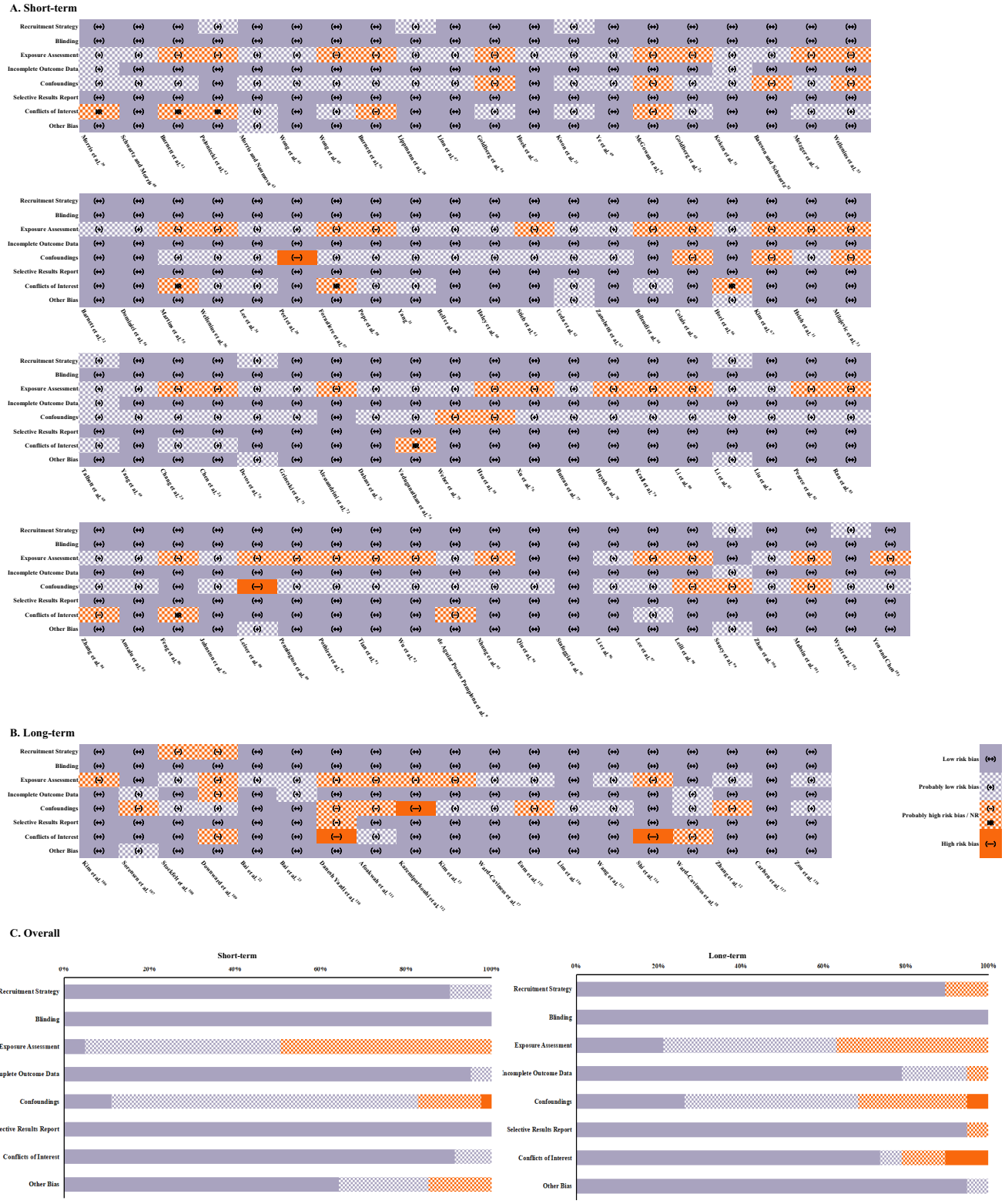

Figure 5. Summary of risk of bias evaluations. The risks of bias for each domain were assessed separately of (A) the short-term exposure and (B) the long-term exposure. (C) The total proportion of each risk level of each domain was calculated. The evaluation of risk of bias was based on the Navigation Guide[14,15] and the detailed criteria were adapted to fit the topic of the review. Determinations for each domain were assigned according to Supplemental Material, "Instructions for Making Risk of Bias Determinations." The light purple grid [labeled with "(++)"] represents the "low" risk of bias; light purple and white intersecting grids [labeled with "(+)"] represent the "probably low" risk of bias; dark orange and white intersecting grids [labeled with "(-)"] represent the "probably high" risk of bias; the dark orange grid [labeled with "(--)"] represents the "high" risk of bias. The original data for the figure plotting are listed in Excel Tables S1 and S2. Note: NR, not reported.

Table 4. Heterogeneity and publication bias by air pollutants and exposure term.

	$PM_{2.5}$ (μg/m^3)	PM_{10} (μg/m^3)	NO_2 (ppb)	SO_2 (ppb)	CO (ppm)	O_3 (ppb)
Short-term exposure						
Studies (*n*)	52	38	34	28	30	30
Estimates (*n*)	65	49	42	38	41	40
Median concentration (IQR)[a]	17.90 (11.07–39.90)	44.99 (30.60–54.85)	23.60 (20.31–31.70)	7.70 (5.29–11.16)	1.09 (0.75–1.52)	28.47 (23.42–41.03)
Range (min–max)[b]	2.90–102.10	17.60–131.50	9.16–77.03	0.92–32.01	0.002–5.60	1.88–95.66
Heterogeneity [I^2 (%)]	97.97	97.36	98.63	96.70	94.28	97.99
Publication bias						
Non-adjusted RR (95% CI)[c]	1.018 (1.011, 1.025)	1.016 (1.011, 1.020)	1.038 (1.019, 1.057)	1.032 (1.017, 1.048)	1.032 (1.023, 1.042)	1.010 (0.998, 1.021)
Adjusted RR (95% CI)[d]	1.020 (1.013, 1.027)	1.017 (1.012, 1.021)	1.042 (1.024, 1.060)	1.032 (1.017, 1.048)	1.032 (1.023, 1.042)	1.020 (1.007, 1.033)
p-Value for Egger's test	0.795	<0.001	<0.001	0.077	0.004	0.079
Long-term exposure						
Studies (*n*)	14	8	10	2	3	5
Estimates (*n*)	15	8	10	2	3	5
Median pollutant concentration (IQR)[a]	10.50 (9.70–20.00)	19.40 (16.44–26.55)	13.04 (10.78–15.60)	7.25 (6.38–8.07)	0.53 (0.35–0.72)	43.63 (25.02–49.32)
Range (min–max)[b]	8.90–52.30	13.00–93.00	6.57–21.40	5.53–8.91	0.17–0.90	6.40–55.00
Heterogeneity [I^2 (%)]	99.95	90.99	99.99	98.78	96.83	99.90
Publication bias						
Non-adjusted RR (95% CI)[c]	1.708 (1.034, 2.821)	1.212 (1.010, 1.454)	1.204 (1.069, 1.356)	3.929 (0.282, 54.798)	1.246 (0.849, 1.830)	1.011 (0.860, 1.187)
Adjusted RR (95% CI)[d]	2.219 (1.294, 3.807)	1.212 (1.010, 1.454)	1.204 (1.069, 1.356)	—	1.246 (0.849, 1.830)	0.925 (0.787 1.087)
p-Value for Egger's test	0.761	—	0.249	—	—	—

Note: The increments of each pollutant are 10 μg/m^3 for $PM_{2.5}$ and PM_{10}, 1 ppm for CO, 10 ppb for SO_2, NO_2 and O_3. —, Not applicable; CI, confidence interval; CO, carbon monoxide; IQR, interquartile range; max, maximum; min, minimum; NO_2, nitrogen dioxide; O_3, ozone; $PM_{2.5}$, particulate matter with the diameter ≤2.5 μm; PM_{10}, particulate matter with the diameter ≤10 μm; RR, relative risk; SO_2, sulfur dioxide.
[a]Median pollutant concentration (IQR) derived from the average daily pollutant concentrations reported in each study.
[b]Range of the average pollutant concentrations across the studies from minimum to maximum.
[c]Risk estimates derived from pooled analysis of studies.
[d]Risk estimates after adjustment for publication bias using the Trim and Fill method.

dysfunction, atherosclerosis, cardiac diastolic dysfunction, left ventricular hypertrophy, and myocardial fibrosis[126,127] and ultimately increase the risk of HF. Besides, animal studies have shown that long-term exposure to $PM_{2.5}$ can lead to an increase in biomarkers of myocardial hypertrophy in mice, leading to ventricular remodeling characterized by myosin heavy chain subtype transformation and fibrogenesis, resulting in consistent changes in the early stage of HF.[126]

Two strengths of our meta-analysis should be highlighted. First, we synthesized the comprehensive evidence to assess the relationship of long-term exposure to particulate and gaseous air pollutants with HF for the first time. Second, by incorporating studies conducted in more countries and regions, we provided a more extensive and evident evaluation for the acute impact of air pollution on HF over a wider range of air pollution concentrations. This filled in the gaps of evidence from the LMICs and regions with high pollution levels.

However, several limitations should be noted. First, there was significant heterogeneity across all pollutants in both short- and long-term exposures. Even so, multiple sensitivity analyses by excluding any single or more specific studies still showed the robustness of our estimations. Publication bias did exist for several pollutants, whereas estimates did not substantially change after adjusting for the bias. Second, owing to the limitation of methods and data, we assessed the impact of only the individual pollutant, not the combined effect. Simultaneous exposure to all pollutants may have synergistic impacts, which were not evaluated in our meta-analysis. Third, evidence from LMICs and gaseous pollutants is sparce, especially regarding long-term exposures. The only study in the LMICs was from China and it included 4,866 patients with HF who were followed up for only 1 y.[114] Compared with particulate matter, the number of studies on gaseous pollutants was less than half, especially for CO and SO_2, which seriously affected the strength of the evidence in the review. Hence, inferences about causality and the strength of the evidence were inevitably limited.

Even so, the true impact of air pollution on HF hospitalization, incidence, and mortality still needs to be further explored in many aspects. First, it is arbitrary to isolate short- and long-term exposures of air pollution, so studies that take both into account are urgently needed in the future. Second, most studies generally assume an approximate linear relationship between pollution concentration and HF. The true dose–response relationship between air pollution and HF needs to be further explored. Third, although the associations of air pollutants with HF have been estimated, they may be more pronounced in patients with HF or other basic diseases that can impair heart function.[17,26,114] Owing to the limitation of existing data, we were unable to accurately estimate the impact of air pollution on patients who have different phenotypes or grades of cardiac insufficiency. Fourth, prolonged cumulative exposure to air pollution seemed to result in a higher risk of HF compared with the risk of myocardial infraction[6] and other CVDs.[128] One possible reason is that HF is the end stage of many heart diseases, especially coronary artery disease.[129] Existing heart problems, such as atrial fibrillation, may also aggravate the prognosis of HF.[129] The deeper cause and possible mechanism deserves further research. Therefore, more high-quality studies are urgently needed to further clarify the cardiovascular effects of air pollution.

In conclusion, the present systematic review and meta-analysis provides compelling evidence for a significant association between $PM_{2.5}$, PM_{10}, NO_2, SO_2, and CO and an increased risk for HF regardless of short- or long-term exposure. Our results reinforce the impact of air pollution on cardiovascular health. Sustained public and environmental policies and actions aimed at controlling air pollution are needed to reduce the burden of HF.

Acknowledgments

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There is no original data to share because all the data we used were extracted from original studies listed in the tables.

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